thereof, or introducting into said cells a DNA sequence encoding said one or more proteins, isoforms, analogs, fragments or derivatives in the form of a suitable vector carrying said sequence, \said vector being capable of effecting the insertion of said sequence into said cells in a way that said sequence is expressed in said cells.

- --33. The method according to claim 32, wherein said treating of said cells comprises introducing into said cells a DNA sequence encoding said GLR protein, isoforms, analogs, fragments or derivatives in the form of suitable vector carrying said sequence, said vector being capable of effecting the insertion of said sequence into said cells in a way that said sequence is expressed in said cells.
- --34. The method according t_0 claim 32, wherein said treating of said cells is by transfection of said cells with a recombinant animal virus vector comprising the steps of:
- (a) constructing a recombinant animal virus vector carrying a sequence encoding a viral \surface protein (ligand) that is capable of binding to a specific cell surface receptor on the surface of said cells to be treated and a second sequence encoding a protein selected from said GILR protein, isoforms, analogs, fragments and derivatives that, when expressed in said cells, is capable of inhibiting apoptosis; and
 - (b) infecting said cells with said vector of (a).
- --35. A method of enhancing apoptosis\in cells by inhibiting the activity of GILR proteins in said cells,

fragments or derivatives thereof, according to claim 14, said treatment being by application of a suitable composition containing said antibodies, said active fragments or derivatives thereof to said cells.

--36. A method of enhancing apoptosis in cells by inhibiting the activity of GILR proteins in said cells, comprising treating said cells with an oligonucleotide sequence encoding an antisense sequence for at least part of the DNA sequence encoding a GILR protein according to claim 1, said oligonucleotide sequence being capable of blocking the expression of the GILR protein.

--37. The method according to claim 36 wherein said oligonucleotide sequence is introduced to said cells via a recombinant animal virus vector comprising the steps of: (a) constructing a recombinant animal virus vector carrying a sequence encoding a viral surface protein (ligand) that is capable of binding to a specific cell surface receptor on the surface of said cells to be treated and a second sequence said oligonucleotide sequence.

--38. A method of enhancing apoptosis for treating tumor cells, HIV-infected cells, or other diseased dells by inhibiting the activity of GILR proteins in said cells, comprising:

(a) constructing a recombinant animal virus vector carrying a sequence encoding a viral surface protein capable

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of binding to a specific\tumor cell surface receptor or HIVinfected cell surface receptor or receptor carried by other diseased cells and a sequence encoding an inactive GILR mutant protein that, when expressed in said tumor, HIV-infected, or other diseased cell, is capable of inhibiting the activity of normal endogenous GILR and enhancing apoptosis in said cells; and

- (b) infecting said tumor or HIV-infected cells or other diseased cells with said vector of (a).
- --39. A method of enhancing apoptosis in cells by inhibiting the activity of GILR $pr\phi$ teins in said cells, comprising applying a ribozyme procedure in which a vector encoding a ribozyme sequence capable of interacting with a cellular mRNA sequence encoding a GILR protein according to claim 11, is introduced into said cell's in a form that permits expression of said ribozyme sequence in said cells, and wherein when said ribozyme sequence $\frac{1}{3}$ expressed in said cells it interacts with said cellular mRNA sequence and cleaves said mRNA sequence resulting in the inhibition ϕ f expression of said GILR protein in said cells.
- --40. A method for enhancing apoptosis in cells by inhibiting the activity of GILR proteins in said cells, comprising introducing into said cells a pept\de that is capable of binding the normal endogenous GILR 1n said cells and inhibiting its activity, thereby enhancing apoptosis .--